Inheritance of RESISTANCE TO ASPARAGUS RUST

Results of recent investigations in Illinois

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INHERITANCE OF RESISTANCE TO *Puccinia asparagi* DC IN *Asparagus officinalis* L.[1]

By P. R. Hepler, A. E. Thompson, and J. P. McCollum[2]

DURING THE PAST TWENTY YEARS severe outbreaks of asparagus rust (*Puccinia asparagi* DC) have occurred in the asparagus-producing areas of Illinois. Before these outbreaks, the disease had apparently been controlled by the use of "resistant" varieties. If so, either the pathogen has mutated, or the host plant has been reselected for susceptibility. The general aim of the present study was to determine whether the host plant had been reselected for susceptibility and to determine specifically: (1) whether the present strains of asparagus are more susceptible than the original Washington varieties; (2) what mode of inheritance controls the degree of resistance; and (3) what effect the pathogen has upon the host plant.

REVIEW OF LITERATURE

The first verified report of asparagus rust in this country was made by Halsted in 1896 (11).[3] He drew attention to this disease by contacting pathologists over the entire country. In the next few years reports of severe rust epiphytotics appeared from Massachusetts (36, 37), Connecticut (38), Maryland (20), Vermont (21), Rhode Island (23), South Carolina (31), Iowa (28), New York (33), Indiana (2), Delaware (8), North Dakota (41), and California (34). Most of these reports emphasized the severity of the disease which destroyed most of the older beds in the Atlantic states (34).

Kahn *et al.* (22) recently described the pathogen, *Puccinia asparagi* DC, and disease in some detail. They concluded that fungicides then available would not control rust, and that development of resistant varieties offers the best opportunity for ultimate control.

[1] This bulletin is based on a thesis submitted by P. R. Hepler to the Graduate College of the University of Illinois in partial fulfillment of the requirements for the Ph.D. degree, 1956. The study was conducted under a cooperative research project of the Divisions of Vegetable Crops and Plant Pathology of the Department of Horticulture and was supported in part by funds authorized by the Research and Marketing Act of 1946.

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[3] These numbers refer to "Literature Cited," pages 45-47.

Halsted (12) noted varietal differences in resistance in the first year the disease was recognized. Observation on eight varieties for seven years are summarized in Table 1 (13, 14, 15, 16, 17, 18). Kinney (23) and Pammel and Hodson (28) noted also that the variety Palmetto and a "French" variety, probably Argenteuil, were less susceptible than other varieties under field conditions. Smith (34) claimed that the variety Palmetto saved the Eastern asparagus industry. He

Table 1. — Averages of Infection of Eight Asparagus Varieties, 1897-1903

Variety	Number of plants	Relative susceptibility[a]
		perct. of infection
Columbian White	120	64
Elmira	180	63
Moore's Cross-bred	60	63
Conover's Colossal	120	62
Barr's Mammoth	120	60
Brunswick	120	58
Palmetto	180	37
Argenteuil	60	35

[a] Calculated from Halsted's annual reports.

noted that where Palmetto and Conover's Colossal were interplanted Colossal was practically exterminated in a few years. Anderson (1) did not find any varietal differences among Palmetto, Argenteuil, Conover's Colossal, and Moore's Cross-bred in South Carolina but did note that growers were switching to Palmetto. Sirrine (33) did not find that Palmetto was more resistant than other varieties on Long Island.

Since the disease was not adequately controlled by either varieties or cultural practices, Norton established a major breeding project in Massachusetts in 1906. This work culminated with the release of two "resistant" varieties, Mary Washington and Martha Washington (26, 27). These and their derivatives are the only varieties used by the asparagus industry today.

Norton conducted permanent variety trials and concluded after several years that a major difference in resistance between varieties existed although no immune plants were found. The American varieties as represented by Conover's Colossal and Moore's Cross-bred were more susceptible than the European varieties such as Argenteuil. Argenteuil was "highly immune" unless located next to an uncut field that provided a large amount of inoculum. As the result of one generation of progeny tests, Norton selected three superior plants, one male and two females. The two Washington varieties thus developed were

described as more highly resistant than Argenteuil, the best variety previously available. The recent experiments at Illinois are in agreement with previous findings in showing that Conover's Colossal, Mammoth Emperor, and Snowhead are very susceptible but that the Washington strains are not sufficiently resistant for commercial production in areas favorable for the development of the disease (22).

Several new asparagus varieties — Eden (10), Viking (30), California 500 (19), Waltham Washington (43), Raritan (32), and Seneca Washington[1] — have recently been released. All of these varieties except Eden were selected from the Washington strains. Eden is a selection from the variety Elmira. Three of these varieties — California 500, Waltham Washington, and Raritan — were selected through a maximum of three generations over a period of 25 years. Except for California 500, rust resistance equal to that of the Washington strains is claimed for all these new varieties, although they were selected primarily for yielding ability and quality. Under field conditions in New Jersey, Raritan and California 500 have been observed to be very susceptible as compared with Washington.[2] In Massachusetts, Paradise is very susceptible and California 500 more susceptible than the Washington strains.[3]

Kahn *et al.* (22) suggested that resistance to rust might be obtained from related species such as *Asparagus sprengeri* Regel, *A. plumosus* Baker, *A. virgatus* Baker, or *A. scandens* var. *deflexus* (Thunb.) Baker. They noted, however, that attempts to obtain interspecific hybrids with *A. officinalis* L. were unsuccessful. Resistance of species in the genus *Asparagus* has recently been summarized by Thompson and Hepler (40), who showed that all of the dioecious species tested are at least as susceptible as *A. officinalis* L. and that several perfect-flowered species were not immune, as had been previously suggested.

One might conclude from numerous studies of rust diseases incited by species of *Puccinia* that in all probability physiologic races of *P. asparagi* should exist. The existence of physiologic races of the pathogen have been investigated at this Station by Kahn *et al.* (22) and Beraha (3, 4). Only the highly susceptible type of reaction (analogous to Type IV of the cereal rust scale) has ever been observed on asparagus (3, 4, 22). The absence of intermediate or aberrant types

[1] Robson Quality Seeds Inc., Hall, New York, 1955.

[2] Personal communication with Dr. N. D. Howard, Green Giant Company, Middletown, Delaware.

[3] Correspondence from Dr. R. E. Young, Massachusetts Agricultural Experiment Station, Waltham, Massachusetts, to A. E. Thompson.

of uredial pustules can be considered as evidence that races of *P. asparagi* are non-existent. Beraha (3, 4) demonstrated that several species of *Allium* and all the horticultural varieties of *Allium cepa* L. tested were susceptible to *P. asparagi.* No differences in reaction were detected among the various species or varieties of *Allium* that would aid in determining races of *P. asparagi.* The only type of resistance thus far isolated from the genus *Asparagus* has been a quantitative difference in the intensity of infection rather than a qualitative difference in reaction. The existence of physiologic races of the pathogen has not been demonstrated, since no apparent differential reaction of the host to the pathogen has been indicated by the type of pustule formed.

MATERIALS AND METHODS

The varieties and sources of seeds used in the varietal trials are given in Table 2. The plants crossed to develop the breeding and genetic populations were selected from the 1951 varietal tests (22) as escapes and highly susceptible plants.

The inoculation chambers employed in 1952 and 1953 were glass-o-net chambers 8 feet long, 4 feet wide, and 2½ feet high. Four DeVilbiss No. 15 atomizers per chamber were used to provide artificial dew. These atomizers used 3 to 4 gallons of distilled water per chamber to create and maintain a dew with intermittent atomization. In 1954 and 1955 a single chamber 26 feet long, 4 feet wide, and 3½ feet high, draped with clear, heavy polyethylene plastic was used for all inoculations (Figs. 1 and 2). A dew of small water droplets (Fig. 3) was developed and maintained through continuous atomization of two humidifying nozzles.[1] About 3.7 liters of distilled water per nozzle was atomized during the 12-hour period of inoculation.

The inoculation methods used were those developed by Kahn *et al.* (22) and modified by Beraha (3). Urediospore inoculum was prepared by scraping heavily infected stems collected from the field and sifting the spores through a No. 80 or No. 100 mesh screen. The spore-host tissue mixture was then diluted 20 to 40 times with talc and dusted on the wet plants. All inoculations were begun in early evening and ended shortly after 8:00 a.m. so as to have minimum greenhouse temperatures during the period of inoculation.

All tests except H and J were conducted with plants two to three months old in 2½-inch clay pots. In Tests H and J the seedling spears

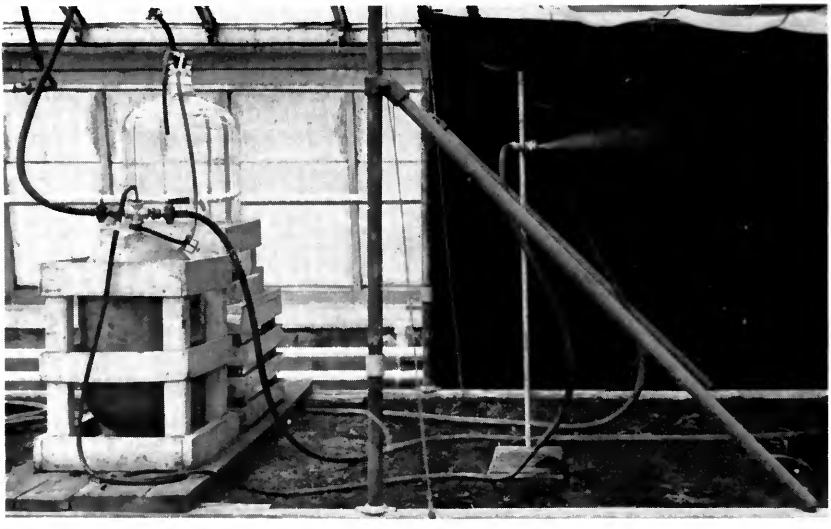
[1] Stainless steel ¼ JNSS pneumatic atomizing spray nozzles — fluid nozzle 2050 and air nozzle 73328. Spraying Systems Company, Bellwood, Illinois.

were tested in flats. Potted plants were randomized when inoculated to minimize both the effect of location within the inoculation chamber and bias while recording infection. The severity of the infection was recorded as either the total number of infection points per plant, or

Table 2. — Accession List and Sources of Asparagus Seed

Accession number	Variety	Stock number, if any	Source of seed
23	Mary Washington	13 B	Vaughan's Seed Co., Chicago, Ill.
25	Paradise	14 B	Vaughan's Seed Co., Chicago, Ill.
27	Viking	.....	Rust-free open-pollinated female plants selected in 1950 at Ill. Agr. Exp. Sta., Urbana
28	Viking	.....	Rust-free open-pollinated female plants selected in 1950 at Ill. Agr. Exp. Sta., Urbana
29	Viking	.....	Rust-free open-pollinated female plants selected in 1950 at Ill. Agr. Exp. Sta., Urbana
37	Conover's Colossal	.....	Hurst & Sons Ltd., Houndsditch, London, England
38	Mammoth Emperor	.....	Carter's Seed Co., Raynes Park, London, England
39	Early Giant Argenteuil	.....	Vilmorin-Andrieux, Paris, France
40	Late Argenteuil	.....	Vilmorin-Andrieux, Paris, France
41	Snowhead	.....	Vilmorin-Andrieux, Paris, France
42	Mary Washington	637/55	Northrup, King & Co., Minneapolis, Minn.
43	Mary Washington	.....	George Pedrick & Sons, Pedricktown, N. J.
44	California 500	10981	Ferry-Morse Seed Co., Detroit, Mich.
45	Mary Washington	70248	Ferry-Morse Seed Co., Detroit, Mich.
46	Mary Washington Giant	.....	L. L. Olds Seed Co., Madison, Wis.
47	Mary Washington	7090	Corneli Seed Co., St. Louis, Mo.
48	Mary Washington	2052	Herbst Brothers, New York, N. Y.
49	California 500	.....	Burrell Seeds, Inc., Rocky Ford, Colo.
50	Mary Washington	.....	Burrell Seeds, Inc., Rocky Ford, Colo.
51	Mary Washington	449	W. Atlee Burpee Co., Philadelphia, Pa.
52	Mary Washington	.....	T. W. Wood & Sons, Richmond, Va.
53	Mary Washington	.....	Robert Buist & Co., Philadelphia, Pa.
54	Mary Washington	.....	H. S. Huber, Pedricktown, N. J.
55	Mary Washington	1401	Barteldes Seed Co., Denver, Colo.
56	Mary Washington	.....	Ritter Seed Co., Bridgetown, N. J.
57	Washington bulk selection	.....	Selection made at Ill. Agr. Exp. Sta., Urbana, from resistant plants received in 1949 from Reg. Veg. Breeding Lab., Charleston, S. C.
58	Washington bulk selection	.....	Selection made at Ill. Agr. Exp. Sta., Urbana, from seven miscellaneous strains received in 1949 from Reg. Veg. Breeding Lab., Charleston, S. C.
59	California 500	39039	F. H. Woodruff & Sons, Milford, Conn.
60	Mary Washington	7-230-3	F. H. Woodruff & Sons, Milford, Conn.
63	Viking	.....	O. J. Robb, Hort. Exp. Sta., Vineland Station, Ontario, Canada
64	Mary Washington	00962	Aggeler & Musser Seed Co., Los Angeles, Calif.
65	Paradise	00963	Aggeler & Musser Seed Co., Los Angeles, Calif.
67	Mary Washington	.....	Eastern States Farmers' Exchange, West Springfield, Mass.
83	Mary Washington resistant selection	45595	F. H. Woodruff & Sons, Milford, Conn.
84	Raritan	.....	L. G. Schermerhorn, N. J. Agr. Exp. Sta., New Brunswick
86	Resistant selection	.....	T. M. Currence, Minn. Agr. Exp. Sta., St. Paul
89	Waltham Washington	.....	R. E. Young, Mass. Agr. Exp. Sta., Waltham Station
90	California 500	39039	F. H. Woodruff & Sons, Milford, Conn.
91	Mary Washington	47039 80	F. H. Woodruff & Sons, Milford, Conn.
92	Martha Washington	3036	Barteldes Seed Co., Denver, Colo.
93	Seneca Washington	Lot #2492	Robson Quality Seeds, Inc., Hall, N. Y.
94	Viking	.....	O. J. Robb, Hort. Exp. Sta., Vineland Station, Ontario, Canada
96	Eden	.....	J. J. Jasmin, Central Exp. Farm, Ottawa, Canada
97	Palmetto	.....	T. W. Wood & Sons, Richmond, Va.
98	Paradise	A-5	Vaughan's Seed Co., Chicago, Ill.
99	Mary Washington (Roberts strain)	.....	Rochelle Asparagus Co., Rochelle, Ill.

General view of the polyethylene draped inoculation chamber used in 1954 and 1955 (Fig. 1)



Details of the humidifying nozzle and the pressure tank for distilled water used in 1954 and 1955. (Fig. 2)

Artificial dew on seedling spears of asparagus. (Fig. 3)

as the number of primary pustules on the axis of the spear. Specific variations in technique are described in the reports of individual experiments.

Percentage of plants infected is given in the results only when it departs significantly from 100 percent. When retested, all escape plants were found to be susceptible. The terms "resistant" and "susceptible" as applied to varieties and experimental populations are relative rather than absolute. Highly susceptible plant material was used as the base for classifying disease reaction. The greatest deviation from the highly susceptible reaction is termed resistance, but the term does not necessarily imply a high level of resistance. Variations in the inoculation technique can produce a very heavy infection on "resistant" plants.

Standard statistical designs and analytical procedures were followed (35). Unless otherwise stated, the original observations were transformed to a logarithmic scale, log $(x + 1)$, prior to analysis. Treatment means are presented as the antilog -1 of the log means. Duncan's multiple range test (9) was used to compare ranked means. According to Duncan's test, "any two means not underscored by the same line are significantly different and any two means underscored by the same line are not significantly different."

RESULTS OF VARIETAL TESTS

The four varietal tests — E, F, G, and H — reported here are a continuation of tests for resistance to asparagus rust reported in 1952 in Illinois Bulletin 559 (22). The purpose of this particular series of tests was: (1) to determine the nature and variability of resistance; and (2) to attempt to isolate a high level of resistance to asparagus rust.

Measure of Resistance of Varieties and Strains Available in 1952 (Test E)

Thirty-two strains of 8 varieties were tested (Table 3). Unpruned plants, selected for uniformity and having about three spears of different ages per plant, were used. All strains were randomized in three glass-o-net chambers in plots of 15 plants each. One hundred and twenty plants per strain were tested. Eight inoculations made on different days were used as replications and counts were made on the entire plant. Data (Table 3) are for only the first inoculation since, when reinoculated, all plants proved susceptible.

Of the 8 varieties, 3 were susceptible and 5 resistant. One strain of each of the susceptible varieties — Snowhead, Mammoth Emperor, and Conover's Colossal — was tested. The following numbers of strains of resistant varieties were tested: Viking, 4; Paradise, 2; California 500, 3; Argenteuil, 2; and Mary Washington, 18.

Highly significant differences were found among the means of the 32 strains and 8 varieties. Major differences between the 3 very susceptible varieties and the 29 strains of the 5 resistant varieties account for most of the statistical difference. When the performance of strains within varieties was measured, only the variability among strains of Washington and Paradise proved statistically significant.

Observation showed that older spears of resistant strains were very seldom infected, while older spears of highly susceptible strains were often quite heavily infected. The youngest spears of all strains were more susceptible than the older spears.

Measure of Resistance of Third Generation Mary Washington Plants (Test F)

Mary Washington plants of known pedigree were compared with these previously tested varieties: California 500; Mary Washington, Paradise, Mammoth Emperor, and Argenteuil. Accession 67 consisted of open-pollinated seed from forty female plants selected for desir-

Table 3. — Percentage of Plants Infected, Mean Number of Pustules per Plant, and Shortest Significant Ranges of Number of Pustules per Plant (Test E)

(Fifteen-plant plots of each of 32 strains of 8 varieties tested in 8 replications)

Accession number	Variety or strain	Percentage of plants infected	Number of pustules per plant	Shortest significant ranges[a]
41	Snowhead	97.5	14.53	
38	Mammoth Emperor	99.2	14.23	
37	Conover's Colossal	93.3	7.04	
29	Viking	86.7	4.58	
42	Mary Washington	92.5	4.45	
50	Mary Washington	92.5	4.06	
27	Viking	91.7	3.83	
25	Paradise	90.0	3.82	
45	Mary Washington	93.3	3.65	
49	California 500	85.8	3.60	
48	Mary Washington	90.0	3.53	
57	Washington bulk selection	92.5	3.44	
47	Mary Washington	85.0	3.33	
44	California 500	90.8	3.28	
28	Viking	90.8	3.13	
39	Early Giant Argenteuil	84.2	3.11	
43	Mary Washington	85.8	3.00	
40	Late Argenteuil	85.8	2.96	
46	Mary Washington Giant	79.2	2.85	
63	Viking	84.2	2.76	
51	Mary Washington	82.5	2.68	
64	Mary Washington	82.5	2.27	
23	Mary Washington	75.8	2.22	
65	Paradise	83.3	2.14	
59	California 500	76.7	2.11	
58	Washington bulk selection	78.3	2.11	
52	Mary Washington	76.7	2.09	
53	Mary Washington	80.0	2.05	
56	Mary Washington	74.2	2.03	
60	Mary Washington	74.2	1.92	
55	Mary Washington	83.3	1.89	
54	Mary Washington	76.7	1.61	

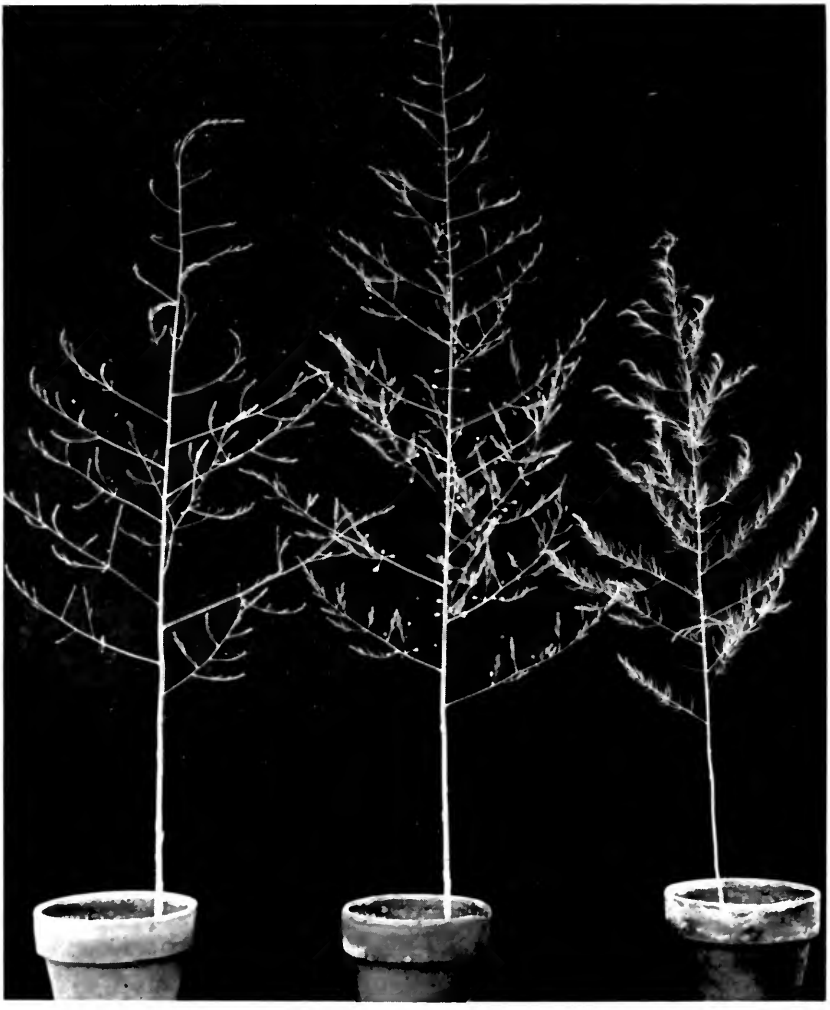
[a] Duncan's test (9), 0.05 level of significance. Number of pustules per plant.

able plant type. The plants were three generations removed from the two original parental plants selected by Norton (27).[1] Ten-week-old plants were pruned to a single spear selected for the most susceptible

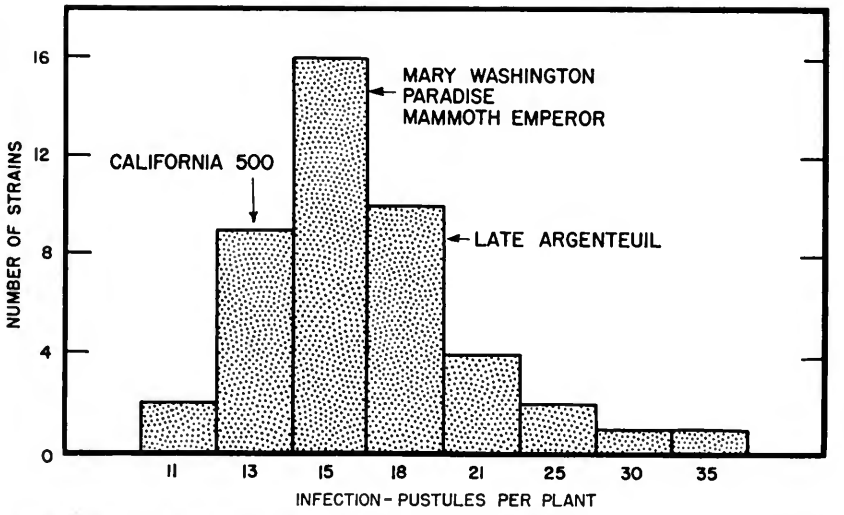
[1] Dr. O. H. Pearson, Eastern States' Farmers' Exchange, supplied information concerning the pedigree. Mr. R. R. Coker, President of Coker's Pedigreed Seed Company, Hartsville, S. C., verified the information.

stage, the 50-percent needle (cladophyll) stage (Fig. 4). Forty-five strains were randomized, four plants per plot in each of four glass-o-net chambers and replicated three times. Infection counts included the entire spear.

A large degree of variability was found within the progenies of the 40 Mary Washington plants, as the data show (Fig. 5). The results are divergent from the expected, since Mammoth Emperor did



Plants (*left to right*) at the 10-, 50-, and 90-percent needle stages. Plants potted in four-inch clay pots. (Fig. 4)



Distribution of 40 Mary Washington F_3 progenies plus California 500 (Accession 49), Mary Washington (Accession 45), Paradise (Accession 25), Mammoth Emperor (Accession 38), and Late Argenteuil (Accession 40) (Test F). (Fig. 5)

not exhibit the proportionately high susceptibility found in Test E and in tests by Kahn *et al.* (22). In two of the three replications, the infection counts included a large number of infected cladophylls which may have obscured some of the differences. The interaction of strains within chambers observed in this experiment was relatively small and indicates that the 45 strains responded similarly throughout the experiment, even though significant differences in the level of infection were found among the four inoculation chambers.

Effect of Age of Spear on Level of Infection of Varieties (Test G)

Because the results of the two previous tests differed, Test G was designed to determine whether there was an interaction between the age of the spear and variety. Five-plant plots of each of eleven strains were randomized within eight replications. Three spear ages per plant were obtained by inoculating when the third spear had reached the 50-percent needle stage. Four replications were inoculated in the polyethylene chamber on each of two days. The fresh weight per plot of five spears of one age was measured to the nearest tenth of a gram. Infection counts were made on the entire spear. Covariance analysis was used to remove the regression of infection on spear weight. The

Table 4. — Mean Number of Pustules and Mean Weight per Spear and Shortest Significant Ranges of Pustules per Spear (Test G)

(Five-plant plots of each of 11 strains of 4 varieties tested in 8 replications; pustules per spear adjusted for differences in spear weight by covariance)

Accession number	Variety or strain, and spear age	Number of pustules per spear	Shortest significant ranges[a]	Weight per spear, grams
83	Mary Washington	5.49		0.59
49	California 500	4.26		0.81
25	Paradise	4.08		0.70
40	Late Argenteuil	3.71		0.76
67-17	Mary Washington	3.33		0.80
67-15	Mary Washington	3.19		0.78
45	Mary Washington	2.95		0.87
67-47	Mary Washington	2.56		0.90
67-23	Mary Washington	1.82		0.80
67-37	Mary Washington	1.81		1.07
67-38	Mary Washington	1.25		0.93
	First spear	1.20		0.36
	Second spear	3.12		0.74
	Third spear	6.25		1.36

[a] Duncan's test (9), 0.05 level of significance. Number of pustules per spear.

six selections from Accession 67 in Test F were selected for high, low, and intermediate susceptibility to rust. A very susceptible strain, Mammoth Emperor, was planted but was not included because the seed did not germinate. Accession 83 was included as a field-resistant selection.

Infection counts and weight of spears showed that differences in both varieties and ages of spears were highly significant (Table 4). Each of the spear ages of all varieties responded similarly to infection and therefore the interaction between varieties and spear ages was not statistically significant. Even though large varietal differences were present, all varieties fell in the resistant class. A lack of differential response between variety and spear age to infection does not preclude such an interaction had a highly susceptible variety been included in the test. The six Mary Washington progenies ranked the same in this test as in Test F except for Accession 67-47 which had the lowest rust reading in Test F.

The highly significant differential reaction of varieties with regard to spear weight at different ages is an indication of the existence of heritable differences in seedling plant vigor. If differences in weight of seedling spears are found to be associated with ultimate yield potential, they should prove valuable in a selection and breeding program, and their measurements could be readily integrated with tests for rust resistance.

Eight Selected Crosses and Recently Released Varieties Compared With Six Standard Strains (Test H)

Fifteen strains, one of which was entered twice, were arranged in two lattice squares of five replications each. The two squares were inoculated separately. The seedling spear was inoculated in one lattice and the second spear in the other because the first inoculation failed. The plants were grown in flats and thinned to a maximum of ten plants per row. The four blocks per replication were arranged as a tier across the polyethylene chamber with two guard rows at each end. The columns in blocks 3 and 4 were arranged as a mirror image of blocks 1 and 2 in order to remove the effect of location found in Test J (page 20). Only the pustules on the primary axis of the spear were counted.

Analysis of variance showed that differences among the means of varieties were highly significant (Table 5). Efficiency of the lattice square over the randomized complete block design was only 11 percent.

Table 5. — Number of Plants Tested, Mean Number of Pustules per Plant, and Shortest Significant Ranges (Test H)
(Fifteen strains, one entered twice, and 10 replications)

Accession or cross number	Variety or cross	Number of plants tested	Number of pustules per plant	Shortest significant ranges[a]
1590	Susceptible cross	83	26.40	
1581	Susceptible cross	91	21.55	
96	Eden	96	15.16	
93	Seneca Washington	95	11.13	
1081	Resistant cross	92	9.65	
1081	Resistant cross	92	9.44	
94	Viking	97	9.40	
91	Mary Washington	98	8.76	
92	Martha Washington	97	8.37	
99	Mary Washington	98	8.06	
97	Palmetto	94	8.04	
89	Waltham Washington	72	7.46	
98	Paradise	76	7.31	
86	Resistant selection	100	6.61	
90	California 500	79	6.56	
84	Raritan	98	5.59	

[a] Duncan's test (9), 0.05 level of significance.

The two susceptible crosses and resistant Cross 1081 were included to provide a check of the breeding material used in Test J. Cross 1081 is the F_1, of Family 4 (Tables 10-11 and 13-14). The performance of Raritan, California 500, and Paradise in this test does not fully agree

with field observations concerning their performance. The validity of the results, however, depends on the assumption that all spears were at a comparable stage of development. Small differences in age of the initial spears can make considerable difference in the level of infection (39). If among resistant varieties spear development occurred at different rates, these varieties may not have been tested at a comparable stage.

Pustules were concentrated at the tips of spears of resistant varieties and more uniformly distributed on spears of susceptible varieties. Major differences between susceptible crosses and resistant strains were very pronounced and conform to the expected results.

RESULTS OF BREEDING TESTS

Measure of Resistance of First Generation Crosses (Test I)

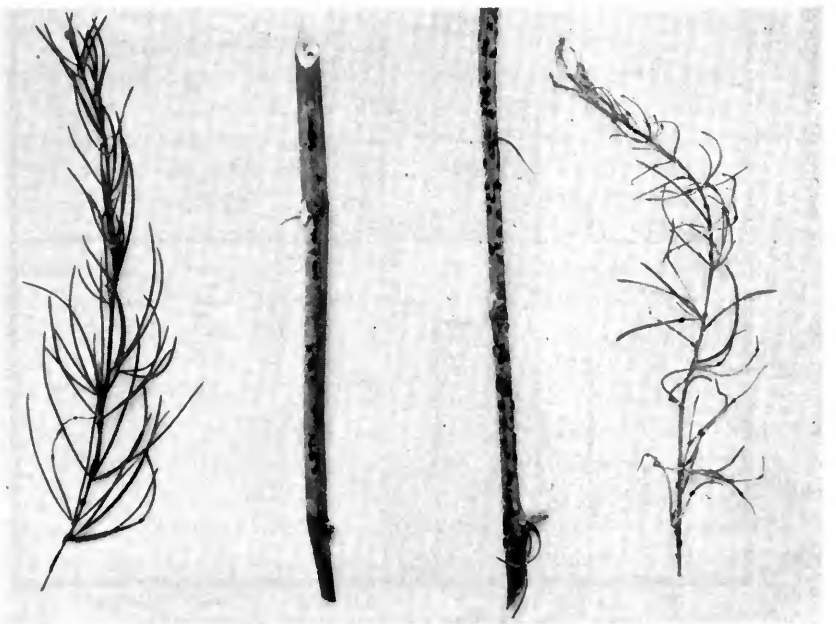
Since variety trials in 1951 (22) indicated considerable differences in susceptibility to *Puccinia asparagi* DC, crosses were made between plants that escaped infection in the 1951 tests (R) and those that had proved highly susceptible (S). (The letters R and S were used mainly as a convenient way of distinguishing the plants as originally selected, since ultimately all plants that escaped infection in 1951 became infected following subsequent inoculations.) Selection was based on the assumption that plants escaping infection in the 1951 tests should have on the average the highest level of resistance. Crosses were made in only three combinations — R × R, R × S, and S × S — because the number of S females was limited. Plots of 4 plants were randomized in four glass-o-net chambers and inoculated at 2-day intervals until ten inoculations had been made. Inoculations were used as replications. The plants were tested at about the third spear stage of development with infection counts made on the entire plant.

Only 9 of the 10 replications were included in the analysis of variance, because in the first replication only 3 of the 596 plants were infected. A breakdown of the analysis of variance showed that within types of crosses the variances for error of the arithmetic data were proportional to the means (Table 6). When the plot totals were transformed to a logarithmic scale, log $(x + 1)$, the variances for error were found to be homogenous. The arc sin $\sqrt{\text{percentage}}$ transformation was used for the percentage of infected plants per plot. (For the general difference in susceptibility between resistant and highly susceptible plants, see Fig. 6. For the distribution of individual-cross means within the three populations, see Fig. 7.)

Table 6. — Means and Associated Error Variances for Number of Pustules per Plant and Percentage of Infected Plants for Three Types of Crosses (Test I)

Type of cross	Number of crosses	Degrees of freedom	Number of pustules per plot				Percentage of infected plants	
			Logarithmic scale		Arithmetic scale			
			Associated error (Log (x+1))	Mean (Antilog −1)	Associated error	Mean	Associated error (Arc sin $\sqrt{\text{percentage}}$)	Mean
R×R.....	90	710	0.0651	2.11	16.42	3.98	407.84	36.6
R×S.....	44	342	0.0674	3.85	38.38	7.28	399.31	59.1
S×S.....	15	111	0.0671	8.24	384.80	18.07	254.84	78.4
Pooled.....	149	1179	0.0682	....	75.36	....	392.90	....

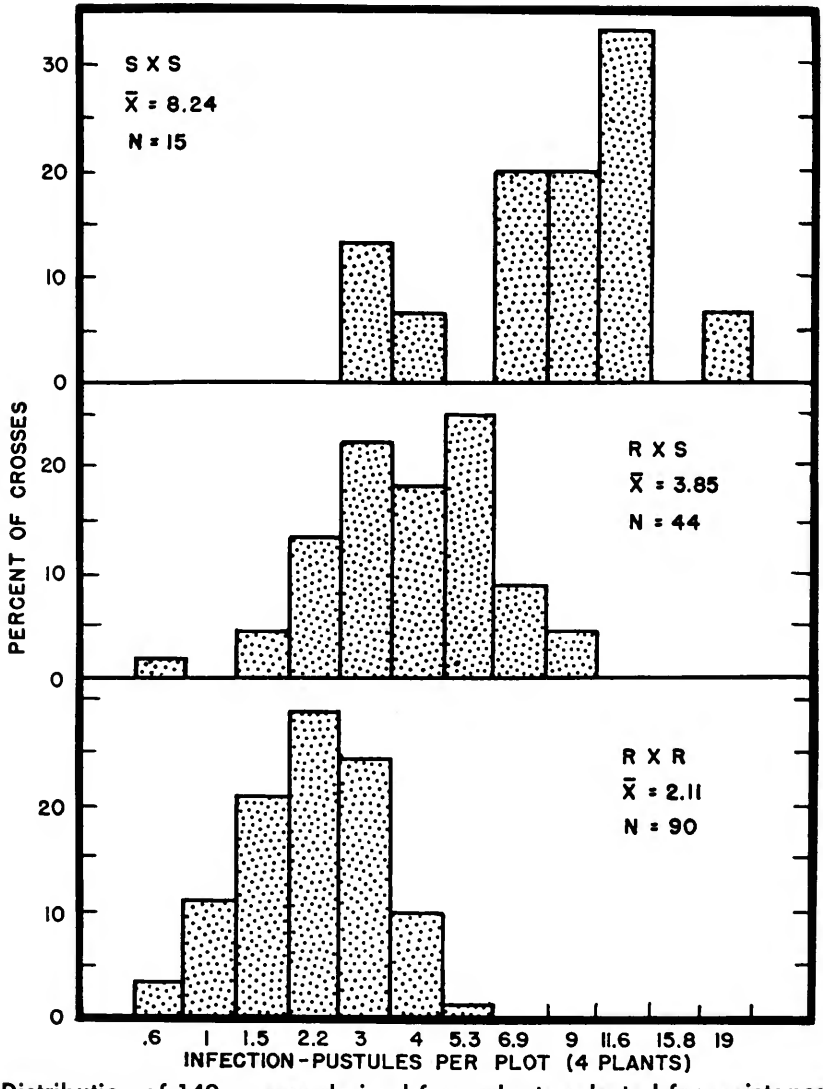
Differences in infection both within and between crosses were highly significant. Within populations of R × R and R × S crosses, parental variety did not account for significant variance. Selection for high susceptibility from within the resistant varieties was not as successful as selection from within highly susceptible varieties. Of 5 crosses using parent plants selected for high susceptibility from resistant varieties,



Typical difference of infection on stem and cladophylls between resistant plant (*left*) and highly susceptible plant (*right*). (Fig. 6)

3 comprise the lower 2 classes of the S × S population. These 3 differ significantly from the 12 remaining S × S crosses, but not from the mean of the R × S crosses.

In 1953, 24 plants each of 25 R × R, 20 R × S, and 10 S × S



Distribution of 149 crosses derived from plants selected for resistance (R) and susceptibility (S) (Test J). **(Fig. 7)**

crosses were retested. The plants, which had been repotted to 4-inch pots, were pruned to a single spear and tested at the 50-percent needle stage. The retest gave similar results at a higher level of infection. The correlation coefficient between the mean values for the 55 crosses in both tests was + 0.787.

Measure of Resistance of Second Generation Crosses (Test J)

Test J was made for two reasons. The first was to test the effectiveness of the second generation of selection and controlled crossing. The second was to evaluate a procedure for testing infection ratings and plant vigor of seedling progenies so as to reduce further the space and time required to test progenies.

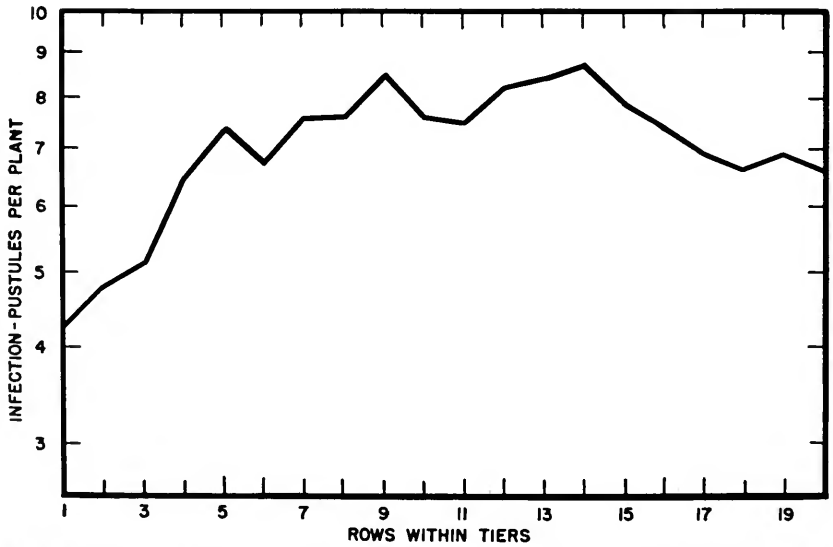
Selections were made between and within R × R and S × S crosses from Test I. About 40 percent of the plants within the crosses selected in Test I were further selected for crossing in Test J. Crossing was limited to making full sib matings and backcrosses to the parents. Twelve families — nine resistant and three susceptible — were selected for Test J. The term family is used here to denote first- and second-generation progenies resulting from two selected plants except for Family 10. Family 10 consisted of 10 of the possible 12 F_1 crosses among seven highly susceptible plants, three females and four males.

The experimental design was a split plot with the twelve families as the first breakdown and the crosses randomized within each family. Each of the six replications was arranged as 10 tiers of 20 plots per tier. A tier consisted of two 16- by 22-inch flats. Fifteen seeds were sown per row. Replications were started at 5-day intervals. Spears commenced to emerge about 10½ days after the seed was sown. Daily germination counts gave percentage of germination and Kotowski's (24) coefficient of the velocity of germination (velocity of germination $= \left(\frac{100}{\text{mean number of days to emergence}}\right)$.

About 21 days after the seed was sowed, each plot was thinned to a maximum of ten plants. The initial spears were measured to the nearest centimeter, and then inoculated in the polyethylene chamber. Nine to twelve days after inoculation, the plants were remeasured. At the same time, primary infection points were counted on the central axis of all spears that were over a minimum of 2 centimeters when they were inoculated. All crosses for which less than 40 plants were measured were eliminated from the analyses. The mean number of plants per cross was 57.

Effect of location in the inoculation chamber on level of infection. The level of infection could be affected by location of plants in the chamber. Differences could arise between tiers, between rows within tiers, or as an interaction between rows and tiers. Plants having inherent differences in susceptibility could conceivably respond differentially to differences in location. The design of the experiment made a complete analysis of the effect of location difficult, since resistant and susceptible crosses were not present in all tiers of any replication. Both resistant and susceptible crosses were present, however, in all 20-tier locations for each replication. A proportional subclass analysis was computed with plant type — resistant vs. susceptible — as the main plots and location within plant type as the subplots. Differences in location within tiers were highly significant, but the two classes of plants responded similarly to location.

The effect of location (Fig. 8) is the result of a drop in the level of infection at the end rows, particularly from the side of the chamber that was opened so that the inoculum could be dusted on the plants. The reduction was very noticeable when the infection readings were made. No variation in distribution of artificial dew was noticeable either when the inoculum was applied or 12 hours later when the



Average number of pustules per plant for the 20 locations within tiers (Test J). (Fig. 8)

plants were washed and removed from the chamber. A correction for this source of error could not be made because of the experimental design used. Variety Test H was designed as a lattice square to reduce this source of variance, but the efficiency of the design was only 11 percent greater than that of a randomized complete block design.

Effect of correlation and regression statistics on precision of infection readings. In order to determine what effect the stage of development of the spear had on the level of infection, simple correlations between pustule counts and supplementary growth measurements were obtained. In general, correlation values for individual families agreed with the pooled values (Table 7). Measurements for seed weight and percentage of germination did not, however, give consistent correlation coefficients and were not studied further. In every family the correlation of the first increment of spear growth to infection was not significant. The velocity of germination was positively correlated with infection in every family and gave values as high at + 0.663. Three variables — velocity of germination, second increment of spear growth, and total spear height — were most highly correlated with infection and were selected for further study.

Multiple covariance was used to correct for regression of infection on velocity of germination, second increment of spear growth, and total spear height. The mean squares and errors of estimate for three families — two resistant and one susceptible — are shown in Table 8. These families were selected for intra-family variability with respect to infection. The F tests (Table 8) show that variability in the crosses of Family 1 were not significant and that within crosses of Families 6 and 18 variability was highly significant. The error variances were not, however, appreciably reduced by the use of the multiple-covariance procedure.

Although the use of multiple covariance did not increase the precision of the information concerning infection, it raised a question as to whether there were differences among average family regressions for infection upon each of the three variables — velocity of germination, second increment of spear growth, and total spear height. Therefore the hypothesis to be tested was that resistance and the supplementary characters are controlled by the same genes in all families. If this proved true, increased resistance to rust could not be expected from crosses between such related families. The errors of estimate (Table 9) indicate that there were no differences between the average family regressions for susceptibility on each of these variables. It may be

Table 7. — Correlation Coefficients Between Rust Infection and Supplementary Growth Measurements (Test J)

Variables	Spear height at inoculation	Second increment of spear growth	Total spear height	Velocity of germination	Seed weight	Percentage of germination
Resistant crosses, n = 659 plots						
Infection, pustules per spear (log (x+1))	+.033	−.370**	−.199**	+.423**	+.005	+.024
Spear height at inoculation			+.767**	+.647**	+.169**	+.335**
Second increment of spear growth			+.690**	−.174**		
Total spear height				+.381**	+.094*	+.364**
Velocity of germination					+.008	+.250**
Seed weight						−.023
Susceptible crosses, n = 330 plots						
Infection, pustules per spear (log (x+1))	+.055	−.337**	−.153**	+.253**	+.109	−.021
Spear height at inoculation			+.701**	+.637**	+.398**	+.227**
Second increment of spear growth			+.738**	−.096		
Total spear height				+.368**	+.444**	+.286**
Velocity of germination					+.049	+.035
Seed weight						+.220**

* Exceeds 0.05 level of significance.
** Exceeds 0.01 level of significance.

Table 8. — Effect of Multiple Covariance of Velocity of Germination, Second Increment of Spear Growth, and Total Spear Height on the Infection Variance (Log (x + 1)) of Three Families (Test J)

Source of variation	Degrees of freedom	Mean square	F test	Multiple correlation (R^2)	Errors of estimate		
					Degrees of freedom	Mean square	F test
Family 1							
Crosses+error	54	0.0117		0.036	51		
Crosses	9	0.0140	1.25		9	0.0158	1.42
Error	45	0.0112		0.076	42	0.0111	
Family 6							
Crosses+error	108	0.0127		0.085	105		
Crosses	18	0.0330	3.79**		18	0.0325	4.22**
Error	90	0.0087		0.139	87	0.0077	
Family 18							
Crosses+error	84	0.0144		0.237	81		
Crosses	14	0.0402	4.32**		14	0.0279	3.49**
Error	70	0.0093		0.179	67	0.0080	

** Exceeds 0.01 level of significance.

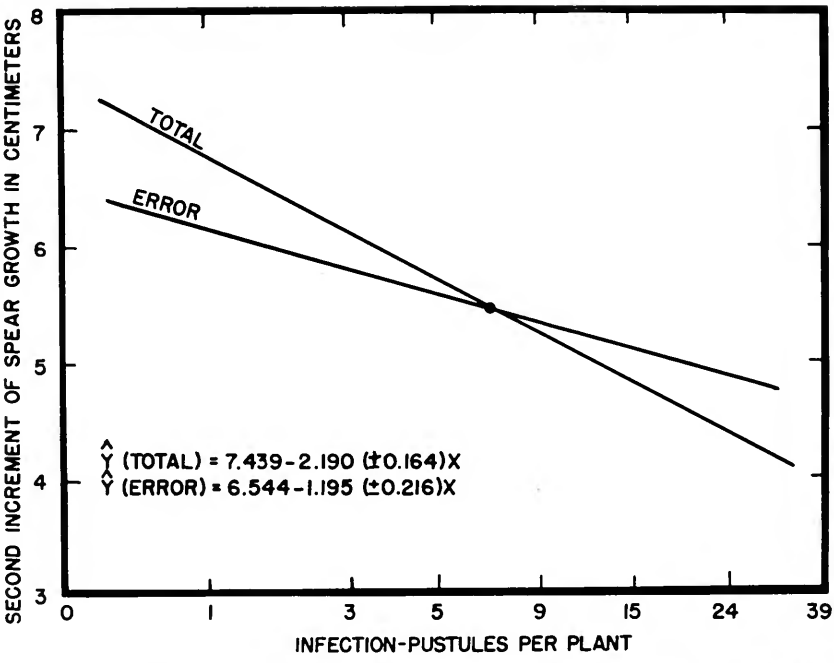
Table 9. — Analysis of Errors of Estimate From Average Regression Within Families for Infection (Log (x + 1)) on Velocity of Germination, Second Increment of Spear Growth, and Total Spear Height (Test J)

Source of variation	Regression coefficient (b)	Degrees of freedom	Sum of squares	Mean square
Infection on velocity of germination				
Deviations from average regression within families	0.185	988	65.9795	.....
Deviations from individual family regressions	.....	977	64.9741	0.067
Differences among family regressions	.....	11	1.0054	0.091 n.s.
Infection on second increment of spear growth				
Deviations from average regression within families	0.069	988	68.1234	.....
Deviations from individual family regressions	.....	977	67.0726	0.069
Differences among family regressions	.....	11	1.0509	0.096 n.s.
Infection on total spear growth				
Deviations from average regression within families	0.029	988	75.2086	.....
Deviations from individual family regressions	.....	977	73.9129	0.076
Differences among family regressions	.....	11	1.2957	0.118 n.s.

n.s. = No significant difference among family regressions.

concluded then that the same genes control susceptibility and the supplementary characters in all families. However, the multiple correlations obtained (Table 8), as well as the simple correlations, indicate that probably less than 15 percent of the variance associated with resistance is thus involved.

When the spears were rated for infection, no pronounced reduction in the length of the infected spears was observed. It was therefore assumed that the second increment of spear growth and final plant height were not dependent on the level of infection. The negative correlation of infection with the second increment of spear growth may thus be examined with infection as the independent variable. This growth period coincides with the incubation period of the fungus plus about 3 days of sporulation. The fungus would be expected to interfere with the development of the spear before the time at which any significant reduction in its photosynthetic surface occurred. Very heavy infections have a severe stunting effect on the affected spears. The level of infection in this experiment, however, would be considered moderate. Since differences among family regressions for either the total



Effect of the level of infection (log (X + 1)) during incubation on second increment of spear growth (measured in centimeters) (Test J). (Fig. 9)

or error terms were not significant, the pooled total and error regression slopes are shown in Fig. 9. The difference between the two slopes was mainly due to the large variance associated with differences between replications in the total regression.

Effect of selection and inbreeding on resistance and supplementary growth characters. The average effect of one generation of full sib mating is to increase inbreeding 25 percent in relation to the parents and to increase it 12½ percent in backcrosses (42). Selection would tend to increase the rate of inbreeding unless the character selected was enhanced by heterosis. In the latter case, inbreeding and selection would reach an equilibrium based on the selection pressure and type of mating system. Variance between families is increased and that within families reduced by inbreeding.

Plants from the nine resistant and two susceptible crosses in Test I were selected only for resistance or susceptibility. Susceptible Family 10 was composed entirely of first generation crosses. Though no specific selection was made for supplementary characteristics, the more precocious plants provided a greater opportunity for making controlled crosses. Since information concerning the parental plants was unavail-

able, random breeding may be assumed for these characteristics. Some bias was introduced into the analyses by eliminating 22 progenies either because their rate of germination was low or slow, or because they produced a high proportion of abnormal first spears. Variances in the measurement of height were further reduced by sampling ten of the more vigorous and uniform plants per plot.

Among crosses within families, the variances for rust infection were smaller than those for the supplementary characters. After two generations of selection for increased resistance, in only 5 of the 9 resistant families was intra-family variability significant.

The percentage of total variance due to the additive genetic differences among crosses within families might also be expressed as heritability. Burton and DeVane (6) working with clones of tall fescue, *Festuca arundinacea,* estimated heritability by separating the variance components in the regular analysis of variance. This method obviates the necessity of using segregating and non-segregating populations for estimates of the genetic and environmental variances. The formula for heritability would thus be:

$$\text{Heritability} = \frac{V_G}{V_G + V_E}$$

$V_E + nV_G$ = the intra-family mean square for crosses
V_E = the intra-family error mean square
V_G = the total intra-family genetic variance
n = the number of replications

Estimates of heritability derived from these equations (Table 10) are for the genetic variability of the selected F_1 parents. To obtain

Table 10. — Estimates of Heritability (Percent) for Resistance and for Supplementary Growth Measurements (Test J)

Family number	Infection (log (x+1))	Spear height at inoculation	Second increment of spear growth	Total spear height	Velocity of germination	Percentage of germination
1	4.0	59.1	10.7	58.2	44.5	33.2
2	10.5	61.8	43.1	61.7	80.9	46.4
3	0.0	55.2	0.0	48.0	48.8	60.3
4	9.7	66.6	23.0	59.6	71.3	39.4
5	17.6	83.2	32.0	73.8	60.7	79.7
6	31.9	59.6	57.3	63.5	70.3	27.5
7	31.5	53.9	0.0	43.9	59.2	0.0
8	13.2	50.3	37.5	51.7	64.1	28.9
9	33.5	25.3	36.5	43.9	35.7	28.0
18	35.7	49.6	44.8	50.2	54.2	0.0
19	15.5	50.2	44.3	46.2	55.2	31.7
Pooled	18.4	57.1	37.1	55.8	62.3	43.5

estimates of the total heritability within families, the mean square for error would have to be partitioned into the components of environmental and genetic variance for plants within plots. The heritability values show that continued selection is not likely to result in increased resistance. Significant improvement in velocity of germination and seedling vigor, however, should be realized from selection.

A more informative test to determine the effect of selection and inbreeding upon resistance is to compare the means of the three generations by a breakdown of the sums of squares for crosses within families (Table 11). In only one resistant family were the means of the backcross and of the F_2 significantly less than the mean of the F_1. In two other families the means of the F_2 were significantly less than the means of the backcross. The pooled effect of the resistant families shows that the level of infection was not significantly reduced by inbreeding and selection within the F_1 generation. All three comparisons were significant in one of the two susceptible families. Susceptibility in the backcross and F_2 generations was reduced in Family 18 rather than increased, though an increase was expected.

The effects of inbreeding on the five measurements of germination

Table 11. — Effect of Selection and Inbreeding on the Resistance of Asparagus to Rust Infection (Test J)

(Number of crosses and average number of pustules per spear for F_1, backcross, and F_2 generations)

Family number	F_1		Backcross		F_2		Comparisons		
	Crosses	Pustules	Crosses	Pustules	Crosses	Pustules	F_1 vs. backcross	F_1 vs. F_2	Backcross vs. F_2
Susceptible families									
18	1	18.57	3	11.40	11	14.08	**	**	**
19	1	10.39	11	10.70	20	10.47			
Resistant families									
5	1	8.24	5	7.05	6	7.39			
6	1	6.70	4	5.43	14	5.84			
8	1	6.25	4	6.02	12	5.46			
2	1	4.96	2	5.47	10	5.54			
4	1	5.18	6	4.62	4	5.05			
3	0	....	1	5.42	11	4.73	**	**	
9	1	5.61	3	4.16	7	4.39	*	*	
7	1	4.65	1	5.33	3	3.99			**
1	1	3.89	3	4.66	6	3.68			**
Resistant families, pooled	8	5.57	29	5.39	73	5.20			

* Exceeds 0.05 level of significance.
** Exceeds 0.01 level of significance.

Table 12. — Effect of Inbreeding on Germination and Seedling Spear Development (Test J)

Variables	Means			Comparisons		
	F_1	Backcross	F_2	F_1 vs. backcross	F_1 vs. F_2	Backcross vs. F_2
Number of crosses....	10	43	104	..	..	..
Spear height, centimeters						
First, at inoculation	10.48	9.32	9.53	**	**	**
Second increment..	5.89	5.33	5.51	**	**	**
Total.....	16.37	14.63	15.03	**	**	**
Velocity of germination.....	7.62	7.40	7.35	**	**	*
Percentage of germination.....	91 2	89.0	89.7	n.s.	n.s.	n.s.

* Exceeds 0.05 level of significance.
** Exceeds 0.01 level of significance.
n.s. = No significant difference.

and growth are given in Table 12. The pooled values of the eleven families containing backcrosses and F_2 crosses are given, since there was no directed selection of F_1 parents for vigor associated with their selection for resistance or susceptibility. The two height increments and the total height of the backcrosses and F_2 crosses were about 10 percent less than those of the F_1 crosses. Results of tests for velocity of germination were similar to those for height, although the percentage of reduction was not as great. Inbreeding did not reduce the percentage of germination consistently or significantly.

The level of rust infection in resistant and susceptible families differed distinctly (Table 13). Only a few crosses overlap between the two populations, but among families within the two populations a considerable number overlapped. The standard errors, however, indicate that family differences were fairly pronounced.

The measurements for velocity of germination and growth of seedling spears showed that differences both between and within the twelve families were very pronounced (Table 14). These characters could be expected to respond to selection since their measurements were relatively precise. No data are available for correlating growth of seedling spears with later growth or yield measurements. Asparagus breeders have thought a cycle of 7 to 10 years was required to produce and test a new generation. Early testing is important, therefore, because it may reduce the time required to produce a new variety.

Table 13. — Family Ranges, Means, and Standard Errors for Number of Pustules per Plant (Test J)

Family number	Number of crosses	Cross means		Family mean	Family mean and standard error (log (x+1))	
		Minimum	Maximum			
Susceptible families						
18	15**	9.25	18.57	13.71	1.169	±0.0102
10	10	8.06	18.57	11.30	1.090	±0.0195
19	32**	7.33	13.68	10.55	1.062	±0.0081
Resistant families						
5	12*	5.39	8.26	7.29	0.920	±0.0102
6	19**	4.51	9.70	5.80	0.832	±0.0088
8	17*	4.37	7.13	5.63	0.822	±0.0086
2	13	4.67	6.71	5.58	0.818	±0.0098
4	11	3.65	6.34	4.82	0.765	±0.0141
3	12	3.74	6.01	4.79	0.763	±0.0157
9	11**	2.82	5.73	4.43	0.734	±0.0122
7	5*	3.22	5.33	4.36	0.730	±0.0150
1	10	3.24	4.99	3.98	0.697	±0.0137

* Difference among crosses exceeds 0.05 level of significance.
** Difference among crosses exceeds 0.01 level of significance.

Table 14. — Family Ranges, Means, and Standard Errors for Supplementary Growth Measurements (Test J)

Family number	Number of crosses	Cross means		Family mean and standard error	
		Minimum	Maximum		
Spear height in centimeters at time of inoculation					
8	17**	10.92	13.37	11.83	±0.064
2	13**	9.72	13.42	11.79	±0.093
7	5**	10.50	12.05	11.13	±0.101
5	12**	7.47	12.62	11.12	±0.070
9	11**	9.53	12.02	10.51	±0.122
4	11**	7.42	10.78	9.32	±0.102
1	10**	6.97	11.47	9.30	±0.159
6	19**	6.43	10.83	8.97	±0.081
10	10**	7.70	10.58	8.86	±0.103
3	12**	7.32	10.18	8.64	±0.105
18	15**	7.07	10.58	8.52	±0.092
19	32**	4.76	9.00	7.50	±0.063
Second increment of spear growth in centimeters					
6	19**	5.27	7.58	6.47	±0.052
7	5	6.15	6.55	6.30	±0.088
8	17**	5.00	7.18	5.95	±0.066
1	10	4.93	6.62	5.84	±0.104
9	11**	5.17	6.73	5.81	±0.068
4	11**	4.92	6.68	5.79	±0.099
18	15**	4.65	6.67	5.61	±0.061
2	13**	4.00	6.33	5.31	±0.073
10	10*	4.73	5.92	5.26	±0.068
3	12	4.67	5.42	5.09	±0.081
5	12**	4.05	5.52	4.98	±0.074
19	32**	3.30	5.90	4.54	±0.048

Table 14. — Concluded

Family number	Number of crosses	Cross means		Family mean and standard error	
		Minimum	Maximum		
Total spear height in centimeters					
8	17**	16.42	20.55	17.78	±0.098
7	5**	16.65	18.33	17.27	±0.130
2	13**	15.50	19.42	17.10	±0.104
9	11**	14.70	18.75	16.32	±0.141
5	12**	12.42	17.72	16.09	±0.104
6	19**	12.97	16.92	15.44	±0.070
1	10**	13.92	17.07	15.15	±0.133
4	11**	12.78	16.47	15.12	±0.118
18	15**	12.70	16.03	14.13	±0.080
10	10**	12.43	16.03	14.13	±0.116
3	12**	11.83	15.45	13.46	±0.126
19	32**	9.60	13.33	12.04	±0.059
Velocity of germination					
8	17**	7.23	8.95	8.37	±0.030
5	12**	7.17	8.87	8.34	±0.043
7	5**	7.70	8.45	8.10	±0.043
2	13**	6.71	8.82	7.86	±0.034
9	11**	7.26	8.25	7.67	±0.044
4	11**	6.32	8.00	7.39	±0.047
3	12**	6.64	7.82	7.16	±0.042
10	10**	6.73	7.42	7.07	±0.042
6	19**	5.95	7.92	6.94	±0.031
1	10**	6.24	7.55	6.93	±0.050
18	15**	6.30	7.35	6.86	±0.030
19	32**	6.13	7.49	6.82	±0.023
Percentage of germination					
18	15	93.3	100.0	96.6	±0.66
10	10	89.8	100.0	94.6	±1.18
5	12**	50.8	100.0	93.0	±0.81
6	19**	77.7	98.8	92.8	±0.72
2	13**	52.3	98.8	91.3	±1.41
7	5	86.7	94.5	91.1	±1.76
8	17**	73.3	97.7	89.6	±0.79
19	32**	72.2	100.0	88.5	±0.67
4	11**	73.3	96.7	88.4	±1.24
9	11**	71.2	97.7	88.0	±1.27
1	10**	68.8	93.3	85.3	±1.10
3	12**	45.7	96.7	78.7	±1.27

** Difference among crosses exceeds 0.01 level of significance.
* Difference among crosses exceeds 0.05 level of significance.

RESULTS OF GENETIC TEST (Test K)

Test I showed that resistance was probably controlled by quantitative genetic factors, since the cross means exhibited continuous variation. Also the F_1 crosses between R and S plants gave a response intermediate between that of R × R and S × S crosses. It was

therefore decided that the inheritance of resistance should be studied more critically and that segregating generations should be obtained for this purpose.

Selection of Parental Material and Experimental Procedure

From Test I three related crosses, giving results typical of the three populations, were selected as parental material. These crosses were —

	Pistillate		Staminate
RR	29-10R	×	45-2R
RS	29-10R	×	41-3S
SS	38-16S	×	41-3S

Random plants from these three crosses were used to obtain six generalized genetic populations (Table 15). Inbred stocks could not be

Table 15. — Genetic Design of Test K

Genetic population	Type of crosses	Number of parental plants		Number of crosses	Number of entries
		Pistillate	Staminate		
P_L	R×R	1	1	1	2
	RR×RR	7	5	8	13
B_L	RR×RS	7	7	9	13
	RS×RR	4	3	4	6
F_1	R×S	1	1	1	2
	S×R	1	1	1	2
	RR×SS	5	4	5	8
	SS×RR	5	5	5	8
F_2	RS×RS	6	6	9	13
B_H	RS×SS	4	4	4	6
	SS×RS	8	6	11	17
P_H	S×S	1	1	1	2
	SS×SS	4	4	6	10
Total	.....	..	..	65	102

obtained because of the dioecious flowering habit of asparagus and its long reproductive cycle. The parental plants, however, were selected from strains giving consistent reactions to tests for resistance and susceptibility. It was therefore assumed that the three types of crosses between RR and SS plants should provide an estimate of the environmental variance component.

Up to 100 seedlings per cross were transplanted to 2½-inch pots. Thirty-seven of 65 crosses consisted of 100 plants each and were

divided into two lots. The 102 entries were randomized within each of eight replications with plots consisting of five plants. The older spears were removed and the third or fourth spear was tested when it reached the 50-percent needle stage. Replications were inoculated in the polyethylene chamber at 2-day intervals from September 3 to 19, 1954. The infected spear was removed 15 to 20 days after inoculation and the primary pustules on the spear axis were counted. Eighteen days after the inoculated spear was removed, yield in grams of fresh weight per plot and number of spears per plant were obtained. Plant survival was recorded December 23. The fifth replication exhibited a pronounced susceptibility gradient from one end of the inoculation chamber and therefore had to be eliminated from the analyses leading to estimates of the number of effective factors and heritability of resistance to rust infection.

Proper Choice of Scale

When any character that exhibits continuous variation is being studied, the choice of a scale adapted to analytical procedures is essential. Whether the effect of the genes is additive or multiplicative may be determined by the type of transformation required to fit the data to an additive model.

Powers (29) working with locule weight in tomatoes found that different scales may be appropriate for different generations in a single experiment, or that different scales may be required by similar plant material in different years. Mather (25) lists two criteria of scaling: the genic effects should be additive and the contribution by the nonheritable agents should be independent of the genotype. Mather gives the following two sets of three equations to test the deviations of the segregating generations from the additive model.

$$A = 2\bar{B}_L - (\bar{P}_L + \bar{F}_1) \qquad V_A = 4V_{\bar{B}_L} + V_{\bar{P}_L} + V_{\bar{F}_1}$$

$$B = 2\bar{B}_H - (\bar{P}_H + \bar{F}_1) \qquad V_B = 4V_{\bar{B}_H} + V_{\bar{P}_H} + V_{\bar{F}_1}$$

$$C = 4\bar{F}_2 - (2\bar{F}_1 + \bar{P}_L + \bar{P}_H) \qquad V_C = 16V_{\bar{F}_2} + 4V_{\bar{F}_1} + V_{\bar{P}_L} + V_{\bar{P}_H}$$

The arithmetic means agreed fairly well with the expected means calculated from the parental populations, while the logarithmic means of the intermediate generations were significantly higher than expected (Table 16). The means of the square root scale, except for the F_2, were also overestimated. The scaling test based on all three nonsegregating populations indicated that the square root scale fitted the additive model for all three segregating populations, while the F_2 of

Table 16. — Observed and Theoretical Means, Standard Errors, and Scaling Test of Three Scales for the Inheritance of Resistance to Rust Infection (Test K)

(Pustules per plant)

Genetic population	Arithmetic scale		Square root scale		Logarithmic scale		Number of plants
	Observed	Theoretical	Observed	Theoretical	Observed	Theoretical	
P_L	30.56±0.647		5.109±0.0541		1.364±0.0096		525
B_L	44.17±0.747	43.34	6.183±0.0534	5.999	1.526±0.0082	1.484	665
F_1	56.25±0.795	56.11	7.057±0.0530	6.889	1.650±0.0072	1.604	700
F_2	53.27±1.056	56.11	6.865±0.0688	6.889	1.627±0.0093	1.604	455
B_H	70.80±0.934	68.88	7.977±0.0537	7.779	1.760±0.0065	1.724	805
P_H	81.65±1.276		8.669±0.0675		1.844±0.0072		420
A	1.53±1.811		0.200±0.1309		0.040±0.0203*		
B	3.70±2.398		0.228±0.1374		0.026±0.0165		
C	−11.63±4.734**		−0.432±0.3072		0.001±0.0417		

$A = 2\overline{B}_L - (\overline{P}_L + \overline{F}_1)$

$B = 2\overline{B}_H - (\overline{P}_H + \overline{F}_1)$

$C = 4\overline{F}_2 - (2\overline{F}_1 + \overline{P}_L + \overline{P}_H)$

* Exceeds 0.05 level of significance.

** Exceeds 0.02 level of significance.

the arithmetic scale and the B_L of the logarithmic scale deviated significantly.

The second criterion of scaling, as previously noted, is independence of the magnitude of the means and variances. Therefore the intra-plot plant variances of the three non-segregating populations, the P_L, F_1, and P_H, were compared. These variances (Table 17) showed that the proportion between the variances and means found with the

Table 17. — Intra-Plot Plant Variances of Six Genetic Populations for Three Scales With Estimates of the Environmental (V_E) and F_2 Genetic Variance ($\frac{1}{2}$ V_D) (Test K)

Source of variance	Degrees of freedom	Scales		
		Arithmetic	Square root	Logarithmic
P_L	420	220.03	1.5372	0.0484
B_L	532	370.70	1.8972	0.0443
F_1	560	442.59	1.9629	0.0361
F_2	364	507.13	2.1527	0.0396
B_H	644	702.61	2.3182	0.0340
P_H	336	684.21	1.9161	0.0216
V_E		448.94	1.8054	0.0354
$\frac{1}{2}V_D$		116.81	0.4760	0.0058

$V_E = (V_{P_L} + V_{F_1} + V_{P_H}) \div 3$

$\frac{1}{2}V_D = \frac{1}{2}(V_{B_L} + V_{F_2} + V_{B_H} - 3V_E)$

arithmetic scale was overcorrected by the logarithmic transformation, while the square root transformation provided the most adequate scale.

These scaling tests would indicate that gene action was more than additive but not geometric.

Estimate of Number of Effective Factors

When the number of effective factors by which two populations differ is estimated, the environmental portion of the variance must be separated from the heritable. With homozygous parental lines, the non-segregating populations, P_L, F_1, and P_H, provide an estimate of the environmental variance, V_E. The backcrosses and F_2 provide information on the additive, V_D, and dominance, V_H, portions of the variance. Mather (25) gives the variance breakdown of the segregating populations as:

$$V_{F_2} = \tfrac{1}{2}V_D + \tfrac{1}{4}V_H + V_E$$

$$V_{B_L} + V_{B_H} = \tfrac{1}{2}V_D + \tfrac{1}{2}V_H + 2V_E$$

The variance due to dominance is thus obtained by subtracting V_{F_2} from $V_{B_L}+V_{B_H}$ after the environmental variance is removed. The degree of dominance is calculated as $\sqrt{\frac{V_H}{V_D}}$. If it is assumed that the dominance variance is 0, then an estimate of the F_2 additive genetic variance, $\frac{1}{2}V_D$, may be obtained from the three segregating populations. The formula is:

$$\tfrac{1}{2}V_D = \tfrac{1}{2}(V_{F_2} + V_{B_L} + V_{B_H} - 3V_E).$$

Estimates of the minimum number of effective factors may be obtained from formulae developed by Castle (7) and by Wright as published by Burton (5). These formulae are based on a model where the genes have equal additive effects. The plus genes are all derived from one parent and the minus genes from the other — no epistasis, no linkage, and independence of the environmental and heritable variances. The formula given by Castle — $n = \frac{(\bar{P}_H - \bar{P}_L)^2}{8\,(V_{F_2} - V_{F_1})}$ — does not take into account the degree of dominance, whereas Wright's formula — $n = \frac{(.75 - h + h^2)\,(\bar{P}_H - \bar{P}_L)^2}{4\,(V_{F_2} - V_{F_1})}$ — where $h = \frac{\bar{F}_1 - \bar{P}_L}{\bar{P}_H - \bar{P}_L}$ — makes an adjustment based on the assumption that the degree of dominance of all plus factors is the same. Departure from these assumptions results in an underestimate of the number of genes determining a character.

The degree of dominance — $\sqrt{\frac{V_H}{V_D}}$ — calculated from variances in Table 17 gives 2.39 for the square root scale. Although a figure larger than 1 would indicate overdominance, the means in Table 16 do not indicate the presence of dominance, since the F_1 and F_2 are on opposite sides of their theoretical values calculated from the parental means of the arithmetic and square root scales.

The analysis of variance of the differences among crosses within the genetic populations (Table 18) shows that the F_2 crosses gave a

Table 18. — Analysis of Variance of Difference in Rust Infection Between and Within Six Genetic Populations (Test K)

Source of variation	Degrees of freedom	Mean squares	
		Square root scale	Logarithmic scale
Replications	6	1832.3585**	29.0458**
Generations	5	853.9394**	15.4840**
Error	30	8.1939	0.2365
Generation:			
P_L Crosses	8	24.6468**	0.7150**
Samples	6	3.3346	0.1014
Error	84	3.7799	0.1259
B_L Crosses	12	25.3082**	0.6053**
Samples	6	3.4372	0.0724
Error	108	6.4536	0.1563
F_1 Crosses	11	29.2854**	0.4808**
Samples	8	3.1489	0.0409
Error	114	4.9835	0.0916
F_2 Crosses	8	3.5589	0.0580
Samples	4	5.2311	0.0738
Error	72	6.9751	0.1215
B_H Crosses	14	15.4977*	0.2494**
Samples	8	8.5960	0.1123
Error	132	7.5192	0.1089
P_H Crosses	6	19.5521	0.1834
Samples	5	16.9046	0.2106
Error	66	10.1872	0.1184

* Exceeds 0.05 level of significance.
** Exceeds 0.01 level of significance.

significantly smaller mean square than was found for the other populations. The estimate of the dominance variance, V_H, is derived from the difference between the F_2 and the sum of the backcross genetic variances. Thus an underestimate of the F_2 variance will lead to an overestimate of the degree of dominance. It would appear then that the F_2 population did not give an adequate estimate of the true genetic variance. This inadequate estimate of variance may have resulted

from the relatively small F_2 population. Lack of precocity in flowering of the RS female plants reduced the size of the F_2 population from that which had been planned.

Divergence of the F_2 variance will also bias estimates of the number of effective factors controlling resistance to rust infection. An underestimate of V_{F_2} will lead to an overestimate of the number of genes. Using the estimated value of $\frac{1}{2}V_D$ (Table 17) based on all three segregating populations would tend to minimize bias introduced by a single

Table 19. — Estimates of Number of Effective Factors and Heritability From the Intra-Plot Plant Variances (Test K)

Measurements	Scales		
	Arithmetic	Square root	Logarithmic
Number of effective factors	2.79	3.34	5.02
Heritability, percent	20.6	20.9	14.2

anormal population. Estimates of the minimum number of effective factors by which the two parental populations differ as calculated by Wright's formula are given in Table 19.

Estimates of Heritability

The estimates of heritability may be based on the intra-plot variances or on the total variability within populations. The first method would employ the general formula for heritability $\frac{V_{F_2}-V_{F_1}}{V_{F_2}}$ where $V_{F_1}=V_E$, and $V_{F_2}=\frac{1}{2}V_D+V_E$ (Table 17). The estimates in Table 19 ignore some of the differences among parental plants as well as the plot error variance component. The within-population

Table 20. — Degrees of Freedom and Variance-Component Breakdown Within Genetic Populations (Test K)

Source of variation	Degrees of freedom	Components of variance
Replications	b−1	
Crosses	c−1	$V_E+V_G+pV_R+pkV_C$
Samples	s	$V_E+V_G+pV_R+pbV_S$
Plot error	(b−1)(c+s−1)	$V_E+V_G+pV_R$
Plants in plots	b(c+s)(p−1)	V_E+V_G
Total	bp(c+s)−1	

b = number of blocks.
c = number of crosses.
s = number of crosses entered twice.
p = number of plants per plot.
k = harmonic mean of the number of plots per cross.

mean squares (Table 18) may be partitioned into their components as listed in Table 20. Then, assuming that V_G is the total additive intra-plot genetic variance and that V_C is the additive genetic variance of the random difference among the within-population parental plants, the total heritability within populations would be: $\frac{V_G+V_C}{V_G+V_C+V_E+V_R+V_S}$. The intra-plot environmental variances for the three segregating populations were calculated from Table 17 as follows:

$$V_E\,(B_L) = \frac{V_{P_L} + V_{F_1}}{2}$$

$$V_E\,(B_H) = \frac{V_{F_1} + V_{P_H}}{2}$$

$$V_E\,(F_2) = \frac{V_{P_L} + V_{F_1} + V_{P_H}}{3}$$

The second equation for heritability gives a more useful estimate of the total heritability within the genetic populations, since selection is based on the differences among crosses within a population as well as among plants within crosses. These estimates (Table 21) are biased, however, since the dominance and epistatic components of variance were not removed from either V_G or V_C.

Table 21. — Total Heritability (Percent) Within Genetic Populations (Test K)

Genetic population	Scales	
	Square root	Logarithmic
P_L	16.7	14.9
B_L	17.4	15.1
F_1	15.3	13.6
F_2	11.1	7.5
B_H	15.3	15.5
P_H	4.4	2.7

A comparison of V_G and V_C in the two backcross populations showed that V_G is about 43 percent of the estimated total additive genetic variance, or about 6.8 percent of the total variance. Selection based on the performance of the individual plants would not be expected to prove effective. These data indicate in part why intra-cross plant selection was ineffectual in Test J (Table 11).

Linkage of Susceptibility and Anthocyanin Development

Most of the qualitative characters noted during the course of these investigations have been lethal chlorophyll-deficient mutants. In addition, a few mutants controlling production of anthocyanin pigment were isolated. One of the four plants, staminate 41-3S, used to develop the genetic populations proved to be homozygous for a dominant gene that inhibits production of anthocyanin (I^p), while the other three parental plants were homozygous for the recessive allele. The genetic populations were two generations removed from these parental plants. Thus 5 of the 6 populations were segregating for purple versus green stems, and 3 of these 5 populations were segregating for both color and resistance. If linkage were present, the greatest difference in resistance between green and purple plants should be found in the F_2, while the least difference should be found in the B_H population. Within the F_1 and P_H no difference in susceptibility should exist between green and purple plants if there was no heritable difference between the two susceptible parental plants with regard to the chromosome carrying the dominant and recessive alleles of the mutant color gene.

Chi square values for the segregation of I^p i^p were not worked out since a good fit for all populations was obvious (Table 22). The

Table 22. — Segregation of a Dominant Inhibitor of Anthocyanin Pigment and Susceptibility to Rust Infection (Test K)

Genetic population	Number of plants	Percentage of I^p —	Expected ratio	Pustules per plant[a]		
				Population mean	Green (I^p —)	Purple (i^pi^p)
P_L	520	0.7	0:1	21.2	....	21.2
B_L	760	48.9	1:1	28.2	30.0	26.5**
F_1	640	50.0	1:1	38.0	36.8	39.3
F_2	520	74.0	3:1	37.6	39.7	32.1**
B_H	920	73.8	3:1	50.4	50.6	49.8
P_H	400	78.5	3:1	55.8	56.1	54.5

[a] Antilog — 1 from log (x + 1) means; 8 replications.
** Difference in infection level between alleles exceeds 0.01 level of significance.

four green plants in the P_L population all appeared in one plot and were probably the result of an error in selecting plants to be inoculated. The comparisons of infection between green and purple plants within 4 of the 5 populations fits the proposed linkage model. The B_H population, however, did not exhibit the expected difference. The relatively small magnitude of difference in the B_L and F_2 would

indicate either a low linkage or resistance governed by several genes, or both. The data indicate that resistance is probably governed by several genes, one of which is linked with I^p.

Effect of Infection on Subsequent Yield

Specific information on the effect of different levels of infection on subsequent plant yield is lacking in the literature. Norton (26) found a correlation of $-.393 \pm .065$ between the level of infection in 1910 and spear diameter in 1911. Data showed reduction of continued spear growth correlated with moderate infection (Fig. 9). Heavy infection severely stunts or kills young spears under both greenhouse and field conditions.

The genetic experiment provided means for estimating the effect of infection on subsequent yield since there were large differences in infection within and between replications. Since the plants were pruned to a single spear and selected for inoculation at the 50-percent needle stage, the photosynthetic area of the plants was limited to the spears tested for resistance to rust. Except for plants with very low infection, few additional spears emerged before spear growth was forced by removing the infected spear for the rust reading. Yield was measured as number of spears per plant and weight of spears per plot (in grams) 18 days after the infected spears were removed. Plant survival was noted approximately 2 months after the yields were obtained. Analyses are based on all eight replications.

All possible simple correlations between infection and yield measurements based on plot totals for each of the six genetic populations

Table 23. — Correlation Coefficients Between Pustules per Plot, Subsequent Yield in Weight and Number of Spears per Plot, and Plant Survival (Test K)

(Number of pustules per plot transformed to square-root scale, yield in grams, number of spears per plant, and number of plants surviving)

Variables	Genetic populations					
	P_L	B_L	F_1	F_2	B_H	P_H
Number of plots	120	152	160	104	184	96
Infection × yield	−.568**	−.560**	−.663**	−.692**	−.676**	−.732**
Infection × spears per plant	−.302**	−.238**	−.432**	−.551**	−.459**	−.517**
Infection × survival	−.200*	−.241**	−.403**	−.413**	−.438**	−.353**
Yield × spears per plant	+.664**	+.629**	+.745**	+.787**	+.620**	+.716**
Yield × survival	+.291**	+.464**	+.527**	+.626**	+.579**	+.542**
Spears per plant × survival	+.174*	+.335**	+.271**	+.506**	+.306**	+.336**

* Exceeds 0.05 level of significance.
** Exceeds 0.01 level of significance.

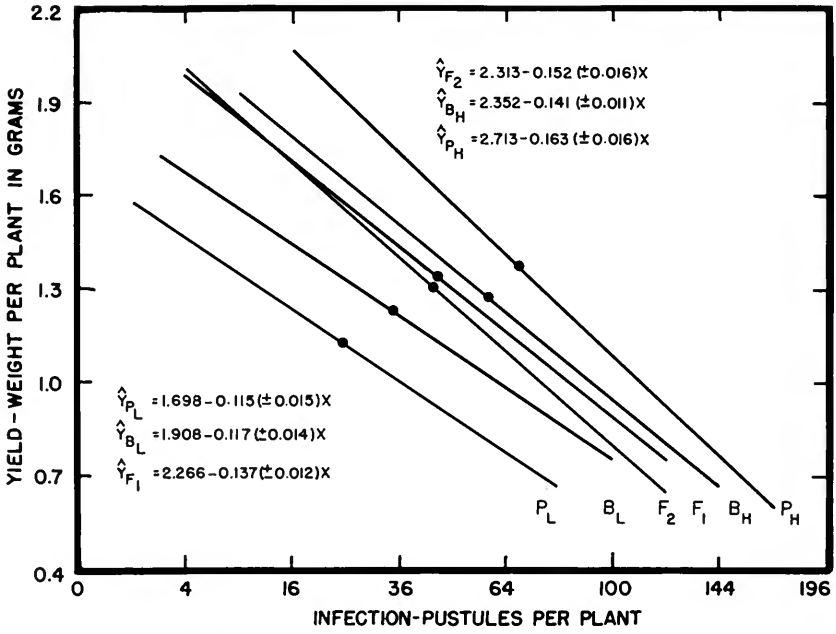
Table 24. — Means and Standard Errors for Number of Pustules per Plant, Yield as Weight and Number of Spears per Plant, and Percentage of Plants Surviving for Six Genetic Populations (Test K)

Genetic population	Number of plants	Number of pustules, square root scale	Yield		Percentage of plants surviving
			Weight per plant, grams	Spears per plant, number	
P_L	600	4.926±0.1685	1.13±0.034	1.22±0.022	70.5±2.11
B_L	760	5.889±0.1840	1.22±0.038	1.25±0.021	54.5±2.27
F_1	800	6.734±0.1985	1.34±0.041	1.30±0.024	63.3±2.14
F_2	520	6.670±0.2187	1.30±0.048	1.32±0.030	58.5±2.56
B_H	920	7.724±0.1866	1.26±0.039	1.30±0.022	52.6±2.12
P_H	480	8.251±0.2787	1.37±0.062	1.39±0.036	52.7±2.64

were calculated (Table 23). (For the means and their standard errors based on the total plot variances, see Table 24.) The correlation coefficients of infection and plant yield in weight (Table 23) indicated that 31 to 54 percent of the yield variance was attributable to differences in levels of infection. The number of spears per plant and survival were also significantly correlated with the level of infection.

It might be expected that populations having a higher susceptibility would give lower yields. However, populations with greater susceptibility gave both higher yields in grams per plant and a higher number of spears per plant. Weight per plant is a function of the number of spears and weight per spear. If weight per spear were calculated, little difference would be found between the six populations. The differences in yield between the populations are therefore largely attributable to the number of spears produced during the 18-day period following the infection rating. Observations on habit of growth corroborate this conclusion, since it was noted that first- and second-generation progeny of staminate plant 41-3S tended to be relatively prolific producers of short spears. Thus the marked reduction in survival is probably due to a combination of high susceptibility to rust and prolific spear production.

Regression analysis failed to show any differences in the slopes of the individual regression lines (Fig. 10). Analysis of the population yields suggested that the differences between the populations were due to the rate at which spears were produced. Thus if all plants were forced until their food reserves were completely exhausted, it might be predicted that the regression line of the P_L population in particular would more nearly coincide with the other lines.



Effect of the level of infection (square root) on subsequent yield (in grams) per plant for the six genetic populations (Test K). (Fig. 10)

Infection levels giving counts of over 50 pustules on the main axis of an 18-inch spear indicated a relatively heavy infection on the side branches and cladophylls. Infection levels at this point or above it reduce yield about 50 percent (Fig. 10). A more specific experiment designed to test these observations would be needed to supply critical information.

DISCUSSION AND CONCLUSIONS

About 55 years ago Halsted (Table 1) observed that asparagus varieties fall into two general levels of resistance to urediospore infection of *Puccinia asparagi* DC. Halsted's observations are confirmed by the experiments reported in this paper with one exception — Test F. This difference in varieties was obvious when the observations on infection were being made, even though wide fluctuations occurred between the infection levels of different inoculations and experiments. Progenies derived from resistant or susceptible varieties consistently performed similarly to their parents.

All the modern varieties except Eden were selected from resistant varieties, and, within the scope of the methods employed in this study, performed like the variety Washington. Eden was released as a resistant selection from Elmira (10). Elmira, however, was described by Halsted as a susceptible variety. In Test H (Table 5) Eden exhibited a significantly higher susceptibility to rust than the resistant varieties. These results suggest that there is a distinct genetic difference between resistant and susceptible varieties and that an adequate level of resistance cannot readily be obtained from highly susceptible varieties.

Highly significant differences were also obtained among resistant strains. These differences were not demonstrated to be discontinuous but formed normally distributed populations. Differences among the resistant varieties were not consistently separable. Unpublished observations by others indicate that under field conditions significant differences in resistance among resistant varieties do occur. These observations suggest that the methods employed in the course of these studies fail to separate specific differences among the resistant varieties.

Under field conditions asparagus plants have spears of several different ages. Differences in the age of spears accumulate on individual plants as the growing season progresses after spring disking or after the cutting season has ended. Asparagus spears exhibit increased resistance with increased age. A rust epiphytotic late in the season will mainly affect the younger spears, while infection early in the growing season often results in most of the spears being heavily infected. Kahn *et al.* (22) observed that fields cut for 60 days escape primary infection from basidiospores. Uncut fields provide a source for primary infection which can initiate epiphytotics in fields after the cutting season has ended.

Without full consideration of cultural practices, field observations could lead to erroneous conclusions about relative varietal resistance. Individual plants of the Washington variety also exhibit marked differences in resistance under field conditions. Few plants in the field are at exactly the same stage of development. The conditions necessary for infection and level of inoculum may also vary from plant to plant. Field observations made on intensity of infection are thus confounded with variations in genetic resistance, variations in stage of development, and with the environment surrounding the plant. Heritability under field conditions would be very low, since with controlled plant and environmental conditions, heritability after one generation of selection was less than 20 percent. Therefore plant selection for rust resistance under field conditions could not be expected to be effectual.

Using artificial inoculation techniques, Beraha (3) found that spears in 2½-inch pots over 20 days old on plants of the variety Snowhead exhibited increased resistance. Thompson and Hepler (39) recently found that seedling spears 7 days after emergence were most susceptible, and those 5 and 9 days after emergence were approximately 18 percent less susceptible. Plants in Test G (Table 4) also showed a very marked increase in resistance with increased spear age. The relative differences in infection among varieties did not change with the age of the spear. Although the more susceptible strains in this test did exhibit greater infection on the older spears, this difference was not great enough to account for the decreased "field resistance" of such resistant varieties as California 500 and Paradise. Testing single spears per plant at the same stage of development should provide an adequate estimate of resistance.

Testing the seedling spear proved to be an economical method for handling a large number of progenies, although the results of varietal Test H raised a question as to the validity of the method. The difference, as expected, between resistant and very susceptible varieties or crosses was distinct in both Tests J and H. The rankings of the resistant varieties in Test H, however, were not as expected.

Observations on the development of the seedling spear indicate that morphological differentiation proceeds rapidly with elongation. The pronounced susceptibility gradient from the tip to the base of the spear also indicates rapid morphological change. Since susceptibility depends on the stage of spear development, plants or strains at different stages of development would not give comparable disease reactions. Attempts to remove the effect of spear age by the use of regression statistics in Test J did not increase the precision of the infection measurements. The validity of the comparisons within families in Test J would not be affected as greatly as the comparisons between families, since the supplementary growth measurements indicated a relatively higher uniformity within than between families.

The third or fourth spear at the 50-percent needle stage did not exhibit as pronounced a differential reaction to infection on different areas of the axis as did the seedling spear. Counting pustules on the side branches and cladophylls, as in Test F, seemed to obscure some of the differences, since the highly susceptible variety Mammoth Emperor gave a relatively low pustule count. Two of the three replications in this experiment gave very high pustule counts. It is possible that spears at the most susceptible age, tested under condi-

tions of maximum infection, will not exhibit heritable differences to rust infection. The variability of the variety Washington was expected since it resulted from only one generation of selection.

It appears doubtful that the Washington variety was highly resistant to rust at the time of its release; a positive statement on the subject, however, is impossible, because nothing is known concerning the relative variability of the pathogen either at the time of its release or at present.

The scaling test in the genetic experiment indicated that the most adequate transformation for adjustment for metrical bias was the square root scale. This test appears to indicate that resistance to rust infection results from genes having more than additive but less than geometric effects. It is obvious, however, that statistical description of these data give little indication of the true mode of gene action. No attempt was made to determine the physiological or morphological basis for resistance.

After the original observations were transformed to an adequate scale, the means and variances indicated that the additive model was adequately fulfilled. Heritability estimates, however, show that the genetic variance components were very small — not over 20 percent. Examination of the variances also shows that the F_2 variance was underestimated, probably because of inadequate sampling. Two of the three populations used to estimate the environmental variance contained a significant variability among crosses within the genetic populations. The intra-plot environmental variance thus may be overestimated, leading to underestimation of the genetic variance and overestimation of the number of genes controlling rust infection.

The equation for estimating the number of genes is based on a model leading to a minimal estimate. The two parent populations differed by a minimum of four to five genes. One of these genes is probably linked with a dominant inhibitor of anthocyanin pigment (I^p). It is difficult to estimate the degree to which the assumptions underlying the formulae for estimating the number of effective factors are fulfilled. The authors consider the equations available for estimating the number of effective factors an inadequate tool. These data do indicate the difficulties involved in breeding for a character modified by environment and a large number of genetic factors. Much research is needed on breeding methods for quantitatively inherited characters, many of which are of great economic importance.

The effect of the rust pathogen on its host has not been subjected

to experimental procedure by previous workers. During incubation the parasite has a pronounced effect on spear elongation (Fig. 9). The yield of heavily infected plants was much reduced after the diseased spear was removed (Fig. 10). Under both field and greenhouse conditions severely infected plants remain semidormant. This mechanism possibly prevents or retards attrition. Heritable differences in bud dormancy would in part explain the much lower plant survival of the genetic populations derived from staminate plant 41-3S.

Sib-mating resistant plants for one generation resulted in an increase in uniformity of the supplementary growth measurements, even though little positive selection was practiced. The relatively high heritability values for the measurements of seedling development suggest that detailed information on plant growth during the first year will result in greater efficiency of selection for yield than is now afforded by extensive yield trials.

In the course of these investigations, over 30,000 plants were tested under artificial conditions. An attempt was made to obtain as broad a collection of germ plasm as possible. Plants that escaped infection in one inoculation were all found to be susceptible when retested. Plants with atypical uredial pustules were never observed. It is thus unlikely that a highly resistant variety will be developed from the germ plasm available for these experiments.

Sib-mating resistant plants for one generation resulted in an increase in uniformity for disease reaction. The average level of resistance might be increased by eliminating the more susceptible germ plasm from the resistant strains. Unless testing procedures are developed that will reduce the large environmental variance, little progress can be expected in selecting for increased resistance to asparagus rust.

SUMMARY

Although over 30,000 plants of *Asparagus officinalis* L. were studied under greenhouse conditions, none were found showing either immunity or a high level of resistance to urediospore infection of *Puccinia asparagi* DC. A major difference in relative susceptibility between varieties was demonstrated. The highly susceptible varieties were Mammoth Emperor, Snowhead, Conover's Colossal, and Eden. The resistant varieties included Mary Washington, Martha Washington, Seneca Washington, Waltham Washington, Argenteuil, Palmetto, Paradise, California 500, Viking, and Raritan. Strains or selections of the resistant varieties exhibited differences which were statistically

significant. Differences among the resistant varieties, however, were not consistently separable.

Crosses among plants selected for resistance (R) and susceptibility (S) exhibited continuous variation with the means of the R × S crosses intermediate between those of the R × R and S × S crosses. Second generation families derived from resistant crosses by sib-mating and backcrossing exhibited pronounced differences between families, but an average heritability of less than 20 percent within families. Plant selection for resistance within R × R crosses and for susceptibility within S × S crosses was ineffectual.

Genetic populations obtained from three related crosses, R × R, R × S, and S × S indicated that the difference between the resistant and highly susceptible populations was determined by at least four or five genes. At least one of the genes for susceptibility was linked with a dominant gene inhibiting anthocyanin development (I^p).

The third or fourth spear on plants 10 to 12 weeks old, inoculated at the 50-percent needle stage, proved the most adequate method for evaluating resistance provided only the primary pustules on the spear axis were counted. A method for inoculating the seedling spear has been described in detail. The disease reaction was shown to be better described by either a square root or logarithmic scale than by the arithmetic scale.

Growth of the infected spear was retarded by the parasite during incubation. Growth of spears following the infected spear was about twice as great from lightly as from heavily infected plants.

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